

## Predictive Value of Cumulative LDL-C Exposure and Uric Acid/HDL-C Ratio for Acute Myocardial Infarction in the Elderly: A Postprint Study

**Authors:** Sun Qinyu, Deng Yifan, Yu Jiling, Cao Gan, Yang Tianxiao, Fang Zhen, Ji Jun, He Shenghu, Zhang Jing, Zhang Jing

**Date:** 2025-09-16T10:24:35+00:00

### Abstract

**Background:** Acute myocardial infarction (AMI) poses a severe threat to the health and quality of life of elderly individuals. Previous studies have identified low-density lipoprotein cholesterol (LDL-C) as an independent risk factor for AMI, and increased uric acid levels are associated with decreased high-density lipoprotein cholesterol (HDL-C) and AMI. However, research on the association between cumulative LDL-C exposure, uric acid/HDL-C ratio (UHR), and the occurrence of AMI in the elderly remains limited.

**Objective:** To investigate the predictive value of cumulative LDL-C exposure, UHR, and their combination for the occurrence of AMI in elderly patients.

**Methods:** We retrospectively enrolled 737 elderly patients diagnosed with AMI at Subei People's Hospital from January 2019 to December 2023 as the experimental group, and 260 elderly patients admitted during the same period but without AMI as the control group. General patient information and laboratory indicators were collected, and cumulative LDL-C exposure and UHR values were calculated. Univariate and multivariate logistic regression analyses were used to screen for independent risk factors for AMI in the elderly. Receiver operating characteristic (ROC) curves were plotted for cumulative LDL-C exposure, UHR, and their combination to predict AMI occurrence in the elderly, and the area under the ROC curve (AUC) was calculated to evaluate the predictive efficacy.

**Results:** The experimental group showed statistically significant differences compared with the control group in gender, smoking history, alcohol abuse history, hypertension history, type 2 diabetes history, BMI, glycosylated hemoglobin A1c, white blood cell count, neutrophil count, monocyte count, triglycerides, total cholesterol, HDL-C, LDL-C, lipoprotein(a), uric acid, cumulative LDL-C exposure, UHR, apolipoprotein A1, and albumin ( $P < 0.05$ ). Multivariate logistic

regression analysis revealed that male gender (OR=3.026, 95%CI=1.769~5.178, P<0.001), type 2 diabetes history (OR=4.834, 95%CI=2.882~8.109, P<0.001), and elevated levels of white blood cell count (OR=1.580, 95%CI=1.368~1.825, P<0.001), LDL-C (OR=3.801, 95%CI=2.712~5.327, P<0.001), cumulative LDL-C exposure (OR=1.898, 95%CI=1.042~3.457, P=0.036), and UHR (OR=10.658, 95%CI=5.147~22.069, P<0.001) were independent risk factors for AMI in the elderly, whereas high levels of HDL-C (OR=0.426, 95%CI=0.184~0.986, P=0.046) and albumin (OR=0.989, 95%CI=0.979~0.999, P=0.031) were independent protective factors. ROC curve analysis showed that the AUCs for cumulative LDL-C exposure and UHR in predicting AMI occurrence in the elderly were 0.726 (95%CI=0.691-0.761) and 0.700 (95%CI=0.663-0.737), respectively, with sensitivities of 0.696 and 0.742, specificities of 0.650 and 0.607, and cutoff values of 214.86 and 317.06, respectively. The combined AUC was 0.813 (95%CI=0.784-0.842), with a sensitivity of 0.723 and specificity of 0.730.

Conclusion: Type 2 diabetes history and elevated levels of white blood cell count, LDL-C, cumulative LDL-C, and UHR are independent risk factors for AMI in the elderly, while high levels of HDL-C and albumin are independent protective factors. Cumulative LDL-C, UHR, and their combination can serve as effective early biomarkers for identifying AMI occurrence in the elderly.

## Full Text

### Study on the Predictive Value of Cumulative LDL-C Exposure and Uric Acid/HDL-C Ratio in Elderly Patients with Acute Myocardial Infarction

SUN Qinyu<sup>1,2,3</sup>, DENG Yifan<sup>1,2,3</sup>, YU Jiling<sup>1,2,3</sup>, CAO Gan<sup>3</sup>, YANG Tianxiao<sup>3</sup>, FANG Zhen<sup>1,3</sup>, JI Jun<sup>1,3</sup>, HE Shenghu<sup>1,3</sup>, ZHANG Jing<sup>1,2,3\*</sup>

1. Department of Cardiology, Northern Jiangsu People's Hospital Affiliated to Yangzhou University, Yangzhou, Jiangsu 225001, China
2. Medical College of Yangzhou University, Yangzhou, Jiangsu 225001, China
3. Department of Cardiology, Northern Jiangsu People's Hospital, Yangzhou, Jiangsu 225001, China

Corresponding author: ZHANG Jing, Chief physician; E-mail: zhangjingyjs@163.com

## Abstract

**Background:** Acute myocardial infarction (AMI) poses a significant threat to the health and quality of life of elderly individuals. Previous studies have identified low-density lipoprotein cholesterol (LDL-C) as an independent risk factor for AMI, while increased uric acid and decreased high-density lipoprotein cholesterol (HDL-C) are also associated with AMI. However, research on the relationship between cumulative LDL-C exposure, the uric acid/HDL-C ratio (UHR), and AMI occurrence in the elderly remains limited.

**Objective:** To investigate the predictive value of cumulative LDL-C exposure, UHR, and their combination for AMI occurrence in elderly patients.

**Methods:** We retrospectively enrolled 737 elderly patients diagnosed with AMI at Northern Jiangsu People's Hospital between January 2019 and December 2023 (experimental group) and 260 elderly patients admitted during the same period without AMI (control group). Baseline demographic data and laboratory parameters were collected, and cumulative LDL-C exposure and UHR values were calculated. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors for AMI in the elderly. Receiver operating characteristic (ROC) curves were plotted for cumulative LDL-C exposure, UHR, and their combined prediction of AMI, with area under the curve (AUC) calculated to assess predictive efficacy.

**Results:** The experimental and control groups showed statistically significant differences in gender, smoking history, alcohol abuse history, hypertension history, type 2 diabetes history, BMI, glycated hemoglobin A1c, white blood cell count, neutrophil count, monocyte count, triglycerides, total cholesterol, HDL-C, LDL-C, lipoprotein(a), uric acid, cumulative LDL-C exposure, UHR, apolipoprotein A1, and albumin ( $P < 0.05$ ). Multivariate logistic regression revealed that male gender (OR=3.026, 95%CI=1.769-5.178,  $P < 0.001$ ), type 2 diabetes history (OR=4.834, 95%CI=2.882-8.109,  $P < 0.001$ ), and elevated levels of white blood cell count (OR=1.580, 95%CI=1.368-1.825,  $P < 0.001$ ), LDL-C (OR=3.801, 95%CI=2.712-5.327,  $P < 0.001$ ), cumulative LDL-C exposure (OR=1.898, 95%CI=1.042-3.457,  $P = 0.036$ ), and UHR (OR=10.658, 95%CI=5.147-22.069,  $P < 0.001$ ) were independent risk factors for elderly AMI. In contrast, high HDL-C (OR=0.426, 95%CI=0.184-0.986,  $P = 0.046$ ) and albumin (OR=0.989, 95%CI=0.979-0.999,  $P = 0.031$ ) were independent protective factors. ROC analysis showed that cumulative LDL-C exposure and UHR predicted elderly AMI with AUCs of 0.726 (95%CI=0.691-0.761) and 0.700 (95%CI=0.663-0.737), sensitivities of 0.696 and 0.742, specificities of 0.650 and 0.607, and cutoff values of 214.86 and 317.06, respectively. The combined model achieved an AUC of 0.813 (95%CI=0.784-0.842) with sensitivity of 0.723 and specificity of 0.730.

**Conclusion:** Type 2 diabetes history and elevated levels of white blood cell count, LDL-C, cumulative LDL-C exposure, and UHR are independent risk factors for elderly AMI, while high HDL-C and albumin are independent protective

factors. Cumulative LDL-C exposure, UHR, and their combination can serve as effective early biomarkers for identifying elderly AMI occurrence.

**Keywords:** Acute myocardial infarction; Cumulative low-density lipoprotein cholesterol exposure; Uric acid to high-density lipoprotein cholesterol ratio; Elderly; Risk assessment

---

## Introduction

Acute myocardial infarction (AMI) represents a critical and severe manifestation of cardiovascular disease in elderly populations, with its pathological basis being acute coronary artery occlusion that leads to sudden interruption of myocardial blood supply and irreversible myocardial cell damage or necrosis [1]. The classic presentation involves sudden, persistent crushing chest pain behind the sternum or in the precordial area, often accompanied by a sense of impending death and dyspnea. Given its high morbidity and mortality, timely and accurate detection is crucial for improving patient outcomes. However, many elderly patients present with atypical symptoms, diminished pain perception, or initial manifestations of heart failure or cardiogenic shock, which significantly increases the risk of misdiagnosis and delays optimal treatment. Therefore, exploring reliable biomarkers for early screening and prediction of AMI in high-risk elderly populations holds important clinical value.

Low-density lipoprotein cholesterol (LDL-C) is a well-established risk factor for cardiovascular disease [2], with substantial evidence demonstrating that elevated circulating LDL-C levels are positively correlated with the formation and progression of coronary atherosclerotic plaques [3]. Currently, monitoring LDL-C levels constitutes a core strategy in dyslipidemia treatment guidelines both domestically and internationally [4]. Dyslipidemia accelerates atherosclerosis, as LDL-C promotes deposition of apolipoprotein B-containing lipid particles in arterial walls by damaging vascular endothelial and smooth muscle cell function, thereby accelerating the atherosclerotic process. As plaque burden progressively increases, it may ultimately lead to acute cardiovascular events [5]. In 2024, Ference et al. [5] proposed the concept of cumulative LDL-C exposure, which can be conveniently calculated using baseline LDL-C values and age. Studies have shown that cumulative LDL-C exposure is positively correlated with the severity of coronary artery disease in patients with acute coronary syndrome (ACS) [6].

Uric acid (UA) is closely associated with cardiovascular disease (CVD). As an effective oxygen scavenger, UA's antioxidant effects can prevent metabolic disorders, but excessive UA promotes LDL-C oxidation and modification, thereby exacerbating atherosclerosis [7]. High-density lipoprotein cholesterol (HDL-C) is an essential component of lipid metabolism and has long been considered protective against coronary atherosclerosis. Research indicates that the combination of low HDL-C and hyperuricemia may exert additive detrimental effects on the

cardiovascular system by synergistically promoting insulin resistance and endothelial oxidative damage [8]. The recently proposed uric acid to HDL-C ratio (UHR) serves as a novel metabolic-inflammatory composite indicator that has been shown to be closely associated with various glucose and lipid metabolism disorders and their prognosis [9]. However, its relationship with AMI occurrence and development in the elderly has not been systematically reported.

Cumulative LDL-C exposure reflects long-term atherosclerotic risk [6], while UHR assesses immediate metabolic disturbance and inflammatory status. Their combination may provide more comprehensive cardiovascular risk prediction: high UA promotes LDL oxidation, while HDL dysfunction exacerbates vascular injury, creating a synergistic pathogenic effect [8]. This study innovatively incorporates both cumulative LDL-C exposure and UHR to explore their combined value, addressing the limitations of single indicators and providing a new strategy for risk stratification. Combined testing can evaluate both chronic cumulative risk and identify acute metabolic abnormalities, holding significant potential for clinical translation.

## Methods

**1.1 Study Population** We retrospectively enrolled 737 elderly patients diagnosed with AMI at Northern Jiangsu People's Hospital from January 2019 to December 2023 (experimental group) and 260 elderly patients admitted during the same period without AMI (control group). The control group included 160 patients with coronary heart disease (CHD) and 100 non-CHD patients. Inclusion criteria were: (1) age  $\geq 60$  years; (2) for the experimental group, AMI diagnosis according to the "Guidelines for the Rapid Diagnosis and Treatment of Acute Coronary Syndrome (2019)" [10] criteria for both ST-elevation and non-ST-elevation myocardial infarction; (3) completion of coronary angiography, with CHD diagnosis defined as  $\geq 50\%$  stenosis in any coronary artery or its major branches; (4) complete clinical data. Exclusion criteria included: (1) regular use of anticoagulant or antiplatelet drugs for CHD secondary prevention; (2) prior use of lipid-lowering medications such as statins or fibrates; (3) non-obstructive AMI due to coronary myocardial bridging or vasospasm; (4) severe hepatic or renal dysfunction; (5) severe heart failure, cardiogenic shock, or other organic heart diseases such as valvular heart disease, malignant arrhythmia, or history of cardiac surgery; (6) hyperthyroidism, severe infection, malignant tumors, autoimmune diseases, or hematological disorders.

Standard definitions were applied: (1) smoking history defined as  $>1$  cigarette daily for  $\geq 6$  months; (2) alcohol abuse defined as  $\geq 20$  g ethanol daily for men and  $\geq 10$  g for women [11]; (3) hypertension diagnosed as systolic blood pressure  $\geq 140$  mmHg (1 mmHg=0.133 kPa) and/or diastolic blood pressure  $\geq 90$  mmHg on three non-consecutive measurements [12]; (4) diabetes diagnosed according to the "Chinese Guidelines for the Prevention and Treatment of Diabetes Mellitus (2024 Edition)" [13]; (5) cumulative LDL-C exposure calculated as baseline LDL-C (mmol/L)  $\times$  age (years) [6]; (6) UHR calculated as

uric acid ( mol/L)/HDL-C (mmol/L).

**1.2 Data Collection** Baseline data were collected through the electronic medical record system, including gender, age, smoking history, alcohol abuse history, hypertension history, and diabetes history. Venous blood samples were collected immediately after admission for hemoglobin, white blood cell count, and other routine blood parameters. After 12 hours of fasting, venous blood was collected the following morning to measure fasting blood glucose and lipid profiles. Left ventricular ejection fraction was precisely assessed using the biplane Simpson method.

**1.3 Statistical Analysis** Statistical analysis was performed using SPSS 27.0 software. The Kolmogorov-Smirnov test was used to assess normal distribution of continuous variables. Normally distributed data were expressed as mean  $\pm$  standard deviation ( $\bar{x}\pm s$ ) and compared between groups using t-tests. Non-normally distributed data were expressed as median (interquartile range) [M(P25, P75)] and compared using rank-sum tests. Categorical data were expressed as proportions and compared using  $\chi^2$  tests. Univariate and multivariate logistic regression analyses were conducted to identify independent risk factors for elderly AMI. ROC curves were plotted to evaluate the predictive value of cumulative LDL-C exposure, UHR, and their combination, with AUC calculated. Statistical significance was defined as  $P<0.05$ .

## Results

**2.1 Comparison of Baseline Characteristics** A total of 997 patients were included (666 males [66.8%] and 331 females [33.2%]), with a median age of 75 (68, 81) years. The experimental and control groups showed significant differences in gender, smoking history, alcohol abuse history, hypertension history, type 2 diabetes history, BMI, glycated hemoglobin A1c, white blood cell count, neutrophil count, monocyte count, triglycerides, total cholesterol, HDL-C, LDL-C, lipoprotein(a), uric acid, cumulative LDL-C exposure, UHR, apolipoprotein A1, and albumin ( $P<0.05$ ). Specifically, the experimental group had higher BMI, glycated hemoglobin A1c, white blood cell count, neutrophil count, monocyte count, triglycerides, total cholesterol, LDL-C, lipoprotein(a), uric acid, cumulative LDL-C exposure, and UHR, but lower HDL-C, apolipoprotein A1, and albumin compared to the control group ( $P<0.05$ ). No significant differences were observed between groups in age, admission systolic/diastolic blood pressure, left ventricular ejection fraction, fasting blood glucose, hemoglobin, lymphocyte count, platelet count, apolipoprotein B, or blood potassium ( $P>0.05$ ).

**2.2 Univariate and Multivariate Logistic Regression Analysis of AMI Risk Factors** Using elderly AMI occurrence as the dependent variable (yes=1, no=0) and gender, smoking history, alcohol abuse history, hypertension history, type 2 diabetes history, BMI, glycated hemoglobin A1c, white blood cell

count, neutrophil count, monocyte count, triglycerides, total cholesterol, LDL-C, HDL-C, lipoprotein(a), apolipoprotein A1, albumin, uric acid, cumulative LDL-C exposure, and UHR (all as continuous variables except where specified) as independent variables, univariate logistic regression identified gender, smoking history, alcohol abuse history, hypertension history, type 2 diabetes history, and elevated BMI, glycated hemoglobin A1c, white blood cell count, monocyte count, LDL-C, lipoprotein(a), uric acid, cumulative LDL-C exposure, and UHR as risk factors, while high HDL-C, apolipoprotein A1, and albumin were protective factors ( $P < 0.05$ ).

Multivariate logistic regression analysis incorporating significant univariate factors revealed that male gender (OR=3.026, 95%CI=1.769-5.178,  $P < 0.001$ ), type 2 diabetes history (OR=4.834, 95%CI=2.882-8.109,  $P < 0.001$ ), and elevated white blood cell count (OR=1.580, 95%CI=1.368-1.825,  $P < 0.001$ ), LDL-C (OR=3.801, 95%CI=2.712-5.327,  $P < 0.001$ ), cumulative LDL-C exposure (OR=1.898, 95%CI=1.042-3.457,  $P = 0.036$ ), and UHR (OR=10.658, 95%CI=5.147-22.069,  $P < 0.001$ ) were independent risk factors for elderly AMI. High HDL-C (OR=0.426, 95%CI=0.184-0.986,  $P = 0.046$ ) and albumin (OR=0.989, 95%CI=0.979-0.999,  $P = 0.031$ ) were independent protective factors.

**2.3 ROC Curve Analysis of Cumulative LDL-C Exposure, UHR, and Their Combination** ROC curve analysis demonstrated that cumulative LDL-C exposure and UHR predicted elderly AMI with AUCs of 0.726 (95%CI=0.691-0.761) and 0.700 (95%CI=0.663-0.737), sensitivities of 0.696 and 0.742, specificities of 0.650 and 0.607, and cutoff values of 214.86 and 317.06, respectively. The combined model achieved an AUC of 0.813 (95%CI=0.784-0.842) with sensitivity of 0.723 and specificity of 0.730 [Figure 1: see original paper].

## Discussion

Cardiovascular disease represents a major global public health challenge, posing a serious threat to human health. According to the latest American Heart Association data, CVD has surpassed cancer as the leading cause of death, with a prevalence of 20% among US adults over 48.6 years [14]. Both mortality and disability rates remain high, particularly in elderly populations. In China, the incidence of coronary atherosclerotic heart disease continues to rise, with AMI being one of its most severe clinical manifestations [15]. The pathophysiological process begins with atherosclerosis progression, characterized by abnormal deposition of lipids, cholesterol, and cellular debris in the coronary artery intima, ultimately leading to severe luminal narrowing or complete occlusion and significantly reduced or interrupted myocardial perfusion [16]. Therefore, investigating the pathogenic mechanisms of elderly AMI, optimizing primary and secondary prevention strategies for coronary heart disease, effectively delaying coronary artery disease progression, and improving long-term clinical outcomes

have become critical scientific challenges in cardiovascular medicine.

Our study confirmed that elevated cumulative LDL-C exposure (OR=1.898, 95%CI=1.042-3.457) and UHR (OR=10.658, 95%CI=5.147-22.069) are independent risk factors for elderly AMI. Dyslipidemia is a key driver of coronary atherosclerosis initiation and progression, and elderly individuals may experience exacerbated lipid metabolism disorders due to age-related decline in hepatic metabolic function, thereby promoting atherosclerosis development. LDL-C, a low-density lipoprotein molecule rich in cholesterol and cholesterol esters, serves as the primary atherogenic lipoprotein that accelerates coronary artery disease by promoting cholesterol deposition in vascular walls [17]. The recently proposed concept of cumulative LDL-C exposure has been utilized as an important biomarker for assessing atherosclerosis progression. This indicator not only reflects cumulative plaque burden but also monitors plaque progression rate and predicts the absolute risk of coronary events at specific time points [5]. Reducing cumulative LDL-C exposure decreases the number of atherogenic lipoproteins trapped within arterial walls, thereby slowing atherosclerosis progression and substantially lowering lifetime risk of atherosclerotic cardiovascular events. Hao et al. [6] demonstrated in an ACS cohort that estimated cumulative LDL-C exposure was significantly positively correlated with coronary artery disease severity and independently predicted major adverse cardiovascular events (MACEs). This suggests that when LDL-C accumulation reaches a threshold level, arterial plaque burden significantly increases the risk of vascular stenosis and plaque rupture. Furthermore, the cumulative effect of LDL-C manifests not only as simple lipid deposition but also through promotion of chronic inflammatory responses and oxidative stress, causing endothelial dysfunction and reduced plaque stability, creating a vicious cycle.

UHR, as the ratio of UA to HDL-C, is now recognized as a novel metabolic and inflammatory marker. Numerous studies have demonstrated strong correlations between UHR and CVD, including atherosclerosis [18], ischemic heart disease [19], hypertension [20], and AMI [8]. Elevated UA can cause atherosclerosis by damaging vascular walls and is closely associated with hypertension, dyslipidemia, and insulin resistance [21]. At the molecular level, UA induces endothelial dysfunction by enhancing endothelial nitric oxide synthase phosphorylation and mediating endoplasmic reticulum stress [9]. When the intracellular environment changes, UA transforms into a pro-oxidant that accelerates coronary atherosclerosis progression. Among various lipoprotein classes, HDL-C is inversely correlated with atherosclerosis incidence. The cholesterol efflux capacity of HDL-C enables it to remove excess cholesterol from macrophages through reverse cholesterol transport and promote its excretion from the body via bile [22]. HDL-C particles in AMI patients exhibit insufficient anti-atherosclerotic activity, which may include impaired macrophage cholesterol efflux and compromised antioxidant, anti-apoptotic, and vasodilatory functions. In a prospective study, researchers found that each 1 mg/dl (0.026 mmol/L) increase in HDL-C level reduced cardiovascular disease risk by 2-3% [23]. Our results showed that each unit increase in UHR increased elderly AMI risk by approximately 10.658-

fold. Yang et al. [24] similarly identified UHR as a risk factor for AMI, with AMI risk increasing as UHR levels rose. Additionally, UHR is associated with CVD prognosis. Li et al. [9] analyzed NHANES data and found UHR to be a strong predictor of cardiovascular mortality in the general population, with the highest UHR quintile showing a hazard ratio of 1.2 (95%CI=1.00-1.45) for CVD death after adjusting for confounders.

This study also confirmed that traditional risk factors such as type 2 diabetes mellitus (T2DM) and LDL-C are significantly associated with elderly AMI, while HDL-C and albumin are independent protective factors. T2DM is a major risk factor for CHD, as T2DM patients are more prone to vascular endothelial dysfunction due to chronic inflammation, oxidative stress, and metabolic disorders, thereby promoting atherosclerosis progression [25]. Inflammatory responses drive coronary artery disease development, and white blood cell count serves as a direct indicator of inflammatory processes. Inflammation causes vascular endothelial injury and recruits immune cells that coordinate early atherosclerotic lesions by releasing pro-inflammatory cytokines, accelerating foam cell formation, causing intraplaque hemorrhage, secreting matrix-degrading enzymes, and promoting lesion progression, ultimately leading to severe coronary disease through various inflammatory cascades [26]. Albumin, the most abundant circulating protein in blood, possesses anti-inflammatory, antioxidant, and antithrombotic properties, with substantial evidence supporting its role as a strong cardiovascular risk predictor across multiple patient populations [27]. In a study of 4,506 individuals followed for 22 years, researchers found that lower serum albumin concentration was positively correlated with AMI risk [28]. After adjusting for risk factors, serum albumin remained an independent protective factor for AMI.

Our ROC curve analysis demonstrated that cumulative LDL-C exposure and UHR predicted elderly AMI with AUCs of 0.726 and 0.700, respectively, indicating good predictive value. These results confirm the important role of these novel markers in AMI development. Notably, combining both markers significantly improved predictive efficacy (AUC=0.813), achieving similar sensitivity (72.3%) while increasing specificity to 73.0%. This combined testing approach demonstrates superior discriminatory ability compared to single markers and provides a more reliable assessment tool for early screening and risk stratification of AMI in elderly populations, holding important clinical application value.

## Conclusion

Cumulative LDL-C exposure and UHR are closely associated with elderly AMI occurrence. This combined indicator has important clinical application value and can provide reliable evidence for early screening of high-risk elderly AMI populations. However, as a single-center retrospective analysis, this study has limitations in sample representativeness and confounding factor control. Future research should expand sample size through multicenter collaboration, extend follow-up duration, perform multiple precise lipid measurements, and incorpo-

rate more potential influencing factors.

### Author Contributions

SUN Qinyu: Conceived the study, designed the research, implemented the study, and wrote the manuscript. DENG Yifan and YU Jiling: Collected and organized data. CAO Gan and YANG Tianxiao: Performed statistical analysis and created figures and tables. FANG Zhen and JI Jun: Revised the manuscript. HE Shenghu and ZHANG Jing: Responsible for quality control and review, overall responsibility for the article, and supervision.

**Funding:** 2025 Jiangsu Province Graduate Research and Practice Innovation Program (SJCX25\_{2401}); 2024 Jiangsu Province Graduate Research and Practice Innovation Program (SJCX24\_{1564})

**Ethical Approval:** This study was approved by the Ethics Committee of Northern Jiangsu People's Hospital (Approval No: 2025ky222). Informed consent was waived for this retrospective study.

**Conflict of Interest:** The authors declare no conflict of interest.

### ORCID:

SUN Qinyu: <https://orcid.org/0009-0002-4896-7168>

ZHANG Jing: <https://orcid.org/0000-0003-3079-3788>

### References

- [1] CHEPELEVA E V. Cell therapy in the treatment of coronary heart disease[J]. *Int J Mol Sci*, 2023, 24(23): 16844. DOI: 10.3390/ijms242316844.
- [2] SILVERMAN M G, FERENC B A, IM K, et al. Association between lowering LDL-C and cardiovascular risk reduction among different therapeutic interventions: a systematic review and meta-analysis[J]. *JAMA*, 2016. DOI: 10.1001/jama.2016.13985.
- [3] PENG K G, YU H L. Characteristic analysis of clinical coronary heart disease and coronary artery disease concerning young and middle-aged male patients[J]. *World J Clin Cases*, 2021, 9(25): 7358-7364. DOI: 10.12998/wjcc.v9.i25.7358.
- [4] BOS D, ARSHI B, VAN DEN BOUWHUIJSEN Q J A, et al. Atherosclerotic carotid plaque composition and incident stroke and coronary events[J]. *J Am Coll Cardiol*, 2021, 77(11): 1426-1435. DOI: 10.1016/j.jacc.2021.01.038.
- [5] FERENC B A, BRAUNWALD E, CATAPANO A L. The LDL cumulative exposure hypothesis: evidence and practical applications[J]. *Nat Rev Cardiol*, 2024, 21(10): 701-716. DOI: 10.1038/s41569-024-01039-5.
- [6] HAO Yichun, CHEN Jing, YAN Shaodi, et al. Correlation between estimated cumulative LDL-C exposure and severity of coronary artery disease and 2-year prognosis in patients with ACS[J]. *Chinese Journal of Cardiology*, 2025, 53(03): 274-280. DOI: 10.3760/cma.j.cn112148-20241031-00661.
- [7] MALOBERTI A, GIANNATTASIO C, BOMBELLI M, et al. Hyperuricemia and risk of cardiovascular outcomes: the experience of the URRAH (uric acid

- right for heart health) project[J]. *High Blood Press Cardiovasc Prev*, 2020, 27(2): 121-128. DOI: 10.1007/s40292-020-00368-z.
- [8] YANG Y, ZHANG J, JIA L, et al. The interaction between uric acid and high-density lipoprotein cholesterol on the prognosis of patients with acute myocardial infarction[J]. *Front Cardiovasc Med*, 2023, 10: 1226108. DOI: 10.3389/fcvm.2023.1226108.
- [9] LI F Q, ZHAO D H, LI Q Y, et al. Uric acid to high-density lipoprotein cholesterol ratio is a novel marker to predict functionally significant coronary artery stenosis[J]. *J Interv Cardiol*, 2022, 2022: 9057832. DOI: 10.1155/2022/9057832.
- [10] Chinese Medical Doctor Association Emergency Medicine Branch, National Health Commission Capacity Building and Continuing Education Center Emergency Medicine Expert Committee, China International Exchange and Promotive Association for Medical and Health Care Emergency Branch. Guidelines for rapid diagnosis and treatment of acute coronary syndrome in emergency department (2019)[J]. *Chinese Journal of Emergency Medicine*, 2019, 28(04): 421-428. DOI: 10.3760/cma.j.issn.1671-0282.2019.04.003.
- [11] SUN Qinyu, DENG Yifan, HE Shenghu, et al. Predictive value of neutrophil-to-lymphocyte ratio, monocyte-to-HDL-C ratio and their combination for contrast-induced nephropathy after emergency percutaneous coronary intervention in patients with acute ST-segment elevation myocardial infarction[J]. *Chinese General Practice*, 2025, 28(15): 1891-1897. DOI: 10.12114/j.issn.1007-9572.2024.0462.
- [12] Chinese Hypertension Prevention and Treatment Guidelines Revision Committee, Hypertension League (China), China International Exchange and Promotive Association for Medical and Health Care Hypertension Branch, et al. Chinese guidelines for the prevention and treatment of hypertension (2024 revision)[J]. *Chinese Journal of Hypertension*, 2024, 32(7): 603-700. DOI: 10.16439/j.issn.1673-7245.2024.07.002.
- [13] Chinese Medical Association Diabetes Branch. Chinese guidelines for the prevention and treatment of diabetes mellitus (2024 edition)[J]. *Chinese Journal of Diabetes Mellitus*, 2025, 17(01): 16-139. DOI: 10.3760/cma.j.cn115791-20241203-00705.
- [14] HOU X Z, WU Q, LV Q Y, et al. Development and external validation of a risk prediction model for depression in patients with coronary heart disease[J]. *J Affect Disord*, 2024, 367: 137-147. DOI: 10.1016/j.jad.2024.08.218.
- [15] HAN Xuexue, ZHANG Tianxing, ZHANG Hao, et al. Study on molecular markers of in-hospital death in acute myocardial infarction[J]. *Chinese Journal of Postgraduates of Medicine*, 2025, 48(01): 66-75. DOI: 10.3760/cma.j.cn115455-20240815-00708.
- [16] ELENDO C, AMAECHI D C, ELENDO T C, et al. Comprehensive review of ST-segment elevation myocardial infarction: Understanding pathophysiology, diagnostic strategies, and current treatment approaches[J]. *Medicine (Baltimore)*, 2023, 102(43): e35687. DOI: 10.1097/MD.00000000000035687.
- [17] AGGARWAL D J, KATHARIYA M G, VERMA D P K. LDL-C, NON-HDL-C and APO-B for cardiovascular risk assessment: Looking for the ideal

- marker[J]. Indian Heart J, 2021, 73(5): 544-548. DOI: 10.1016/j.ihj.2021.07.013.
- [18] AHARI R K, SAHRANAVARD T, MANSOORI A, et al. Association of atherosclerosis indices, serum uric acid to high-density lipoprotein cholesterol ratio and triglycerides-glucose index with hypertension: a gender-disaggregated analysis[J]. J Clin Hypertens (Greenwich), 2024, 26(6): 645-655. DOI: 10.1111/jch.14829.
- [19] PARK B, JUNG D H, LEE Y J. Predictive value of serum uric acid to HDL cholesterol ratio for incident ischemic heart disease in non-diabetic Koreans[J]. Biomedicine, 2022, 10(6): 1422. DOI: 10.3390/biomedicine10061422.
- [20] AKTAS G, KHALID A, KURTKULAGI O, et al. Poorly controlled hypertension is associated with elevated serum uric acid to HDL-cholesterol ratio: a cross-sectional cohort study[J]. Postgrad Med, 2022, 134(3): 297-302. DOI: 10.1080/00325481.2022.2039007.
- [21] HAN X X, TAN X, LIU M Y, et al. Association between serum uric acid/high-density lipoprotein cholesterol ratio and hypertension among reproductive-aged women[J]. J Health Popul Nutr, 2023, 42(1): 122. DOI: 10.1186/s41043-023-00394-5.
- [22] THAKKAR H, VINCENT V, ROY A, et al. HDL functions and their interaction in patients with ST elevation myocardial infarction: a case control study[J]. Lipids Health Dis, 2020, 19(1): 75. DOI: 10.1186/s12944-020-01260-4.
- [23] POTÌ F, SIMONI M, NOFER J R. Atheroprotective role of high-density lipoprotein (HDL)-associated sphingosine-1-phosphate (S1P)[J]. Cardiovasc Res, 2014, 103(3): 395-404. DOI: 10.1093/cvr/cvu136.
- [24] YANG F, WANG J R, WU S L, et al. Effect of cumulative uric acid to high-density lipoprotein cholesterol ratio on myocardial infarction in prospective cohorts[J]. Curr Probl Cardiol, 2025, 50(1): 102889. DOI: 10.1016/j.cpcardiol.2024.102889.
- [25] WU X S, QIU W P, YANG H C, et al. Associations of the triglyceride-glucose index and atherogenic index of plasma with the severity of new-onset coronary artery disease in different glucose metabolic states[J]. Cardiovasc Diabetol, 2024, 23(1): 76. DOI: 10.1186/s12933-024-02178-0.
- [26] ATTIQ A, AFZAL S, AHMAD W, et al. Hegemony of inflammation in atherosclerosis and coronary artery disease[J]. Eur J Pharmacol, 2024, 966: 176338. DOI: 10.1016/j.ejphar.2024.176338.
- [27] MANOLIS A A, MANOLIS T A, MELITA H, et al. Low serum albumin: a neglected predictor in patients with cardiovascular disease[J]. Eur J Intern Med, 2022, 102: 24-39. DOI: 10.1016/j.ejim.2022.05.004.
- [28] DJOUSSÉ L, ROTHMAN K J, CUPPLES L A, et al. Serum albumin and risk of myocardial infarction and all-cause mortality in the Framingham Offspring Study[J]. Circulation, 2002, 106(23): 2919-2924. DOI: 10.1161/01.cir.0000042673.07632.76.

---

*Received: June 22, 2025; Revised: July 30, 2025*  
*Edited by: LI Weixia*

*Note: Figure translations are in progress. See original paper for figures.*

*Source: ChinaXiv – Machine translation. Verify with original.*